Right ventricular myocardial infarction

Authors
Thomas Levin, MD
James A Goldstein, MD

Section Editors
Freek Verheugt, MD, FACC, FESC
Donald Cutlip, MD

Deputy Editor
Gordon M Saperia, MD, FACC

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INTRODUCTION — Acute myocardial infarction (MI) involving only the right ventricle is an uncommon event. More often, right ventricular infarction (RVMI) is associated with acute ST-elevation myocardial infarction of the inferior wall of the left ventricle, and occurs in 30 to 50 percent of such cases [1-6].

RVMI is associated with higher in-hospital morbidity and mortality compared to patients with a similar infarction territory in the left ventricle but that does not involve the right ventricle. Poor outcome is usually related to profound hemodynamic and electrical complications, which occur in approximately 50 percent of affected individuals [1-9]. However, long-term prognosis is generally good for those who survive the event.

This topic will discuss the diagnosis and management of patients with RVMI. The general approach to patients with MI is found elsewhere. (See "Overview of the acute management of unstable angina and non-ST elevation myocardial infarction" and "Overview of the acute management of ST elevation myocardial infarction" and "Overview of the non-acute management of ST elevation myocardial infarction" and "Overview of the non-acute management of unstable angina and non-ST elevation myocardial infarction".)

BASIC CONCEPTS

Definitions — The following terms that are used in this topic are defined as follows:

- **Stroke volume** is the amount of blood pumped with each beat. It is influenced by preload, afterload, and contractility. (See "Pathophysiology of heart failure: Left ventricular pressure-volume relationships", section on 'Normal left ventricular pressure-volume relationship'.)
- **Preload** is the volume of blood within the left or right ventricle at the end if diastole (the filling period). Up to a point, increasing preload leads to a rise in stroke volume. (See "Pathophysiology of heart failure: Left ventricular pressure-volume relationships", section on 'Preload'.)
- **Afterload** is the resistance to forward flow from the right or left ventricle (figure 1). In patients with ventricular dysfunction, increasing afterload leads to a fall in stroke volume. (See "Pathophysiology of heart failure: Left ventricular pressure-volume relationships", section on 'Afterload'.)
- **Contractility** is the strength of contraction (at any ventricular size) of the myocardial muscle. Up to a point, increasing contractility increases stroke volume. (See "Pathophysiology of heart failure: Left ventricular pressure-volume relationships", section on 'Contractility'.)
- **Cardiac output** is the amount of blood pumped per minute and is the product of the stroke volume and heart rate.

Right ventricular infarction versus ischemia — The term right ventricular (RV) "infarction" is, to an extent, a misnomer, as most cases of acute RV ischemic dysfunction appear to represent predominantly viable myocardium. This is in marked contrast to the effects of ischemia and reperfusion on the left ventricle in which prolonged ischemia often leads to infarction [10,11]. The following observations support ischemia, rather than infarction, as the mechanism by which adverse outcomes are precipitated:

- Right ventricular function improves in the majority of patients with right ventricular infarction, including those who are not reperfused [7,8,12-14]. (See 'Long-term prognosis' below.) There are several factors that make the right ventricle less susceptible to infarction:
  - Compared to the left ventricle, oxygen demand is significantly lower in the right ventricle because of its much smaller muscle mass and lower afterload [15,16].
  - Coronary perfusion in the right ventricle occurs in both systole and diastole [16,17].
  - There is more extensive collateral flow from left to right coronary arteries [18].
- The right ventricle may also be protected from infarction to a greater degree than the left ventricle by ischemic preconditioning [19]. (See "Clinical implications of ischemic preconditioning".)
- Chronic right heart failure attributable to right ventricular infarction (RVMI) is rare.

Site of the culprit lesion — In most individuals, the majority of the right ventricle is supplied by the right coronary artery through RV marginal branches. Thus, the majority of RV infarcts result from occlusion of the right coronary artery proximal to the origin of the major RV branches [1-3,5,20,21].

However, in patients with a left dominant system, which occurs in about 15 percent of the general population (or those with a chronically occluded proximal right coronary artery and significant collateral blood flow from either the left anterior descending or circumflex coronary artery), more than 50 percent of the right ventricle can be supplied by the left coronary circulation.

Hemodynamic consequences — The hemodynamic consequences of RVMI depend on the extent of RV free wall dysfunction; the presence of concomitant right atrial ischemia (resulting from very proximal occlusions), which leads to underfilling of the RV; and the extent of simultaneous left ventricular impairment. Clinically evident hemodynamic manifestations are seen in less than 50 percent of affected patients [22]. It should be kept in mind that in patients with RVMI, hemodynamic compromise may be due to RV dysfunction, left ventricular (LV) dysfunction, or a combination of these. In some cases, the relative contribution of the right and left ventricles may be uncertain and hemodynamic monitoring may be required both to understand the pathophysiology and to guide treatment. (See 'Hemodynamic monitoring' below.)

Proximal occlusion of the right coronary artery compromises RV free wall perfusion, leading to dyskinesis and depressed global RV performance [2,6,19,20]. This leads to a fall in the delivery of blood to the left ventricle and decreased systemic cardiac output despite intact LV systolic performance. RV (and LV) diastolic dysfunction also contributes to hemodynamic compromise [23]. The ischemic RV is stiff and dilated early in diastole, impeding inflow from the right atrium and leading to rapid diastolic pressure elevation. RV dilatation and elevated diastolic pressure shift the interventricular septum toward the volume deprived LV, further impairing LV compliance and filling. Abrupt RV dilatation within the non-compliant pericardium elevates intrapericardial pressure, the resultant constraint further intensifying septal-mediated diastolic ventricular interactions and thereby impairing both RV and LV compliance and filling.

Other factors that may worsen the hemodynamic profile of patients with RVMI include:

- Right atrial ischemia can impair right atrial function, worsening the hemodynamic changes [21,24].This occurs when the culprit right coronary artery lesion is proximal to the right atrial branches.
- Depression of LV function (for example, from prior MI) may exacerbate hemodynamic compromise for any degree of ischemic RV dysfunction. This effect is attributable not only due to loss of LV power, but also due to the fact that under conditions of severely depressed RV free wall contraction, RV performance is dependent on LV-septal contraction, which is generally abnormal with prior LV systolic dysfunction.
- Tricuspid valve regurgitation, caused by ischemia to the papillary muscle or by dilatation of the tricuspid annulus.
- Mechanical complications of MI, such as ventricular septal rupture [2,25]. The left-to-right shunting further reduces left ventricular output and exacerbates the right ventricular dysfunction. (See "Mechanical complications of acute myocardial infarction".)

Electrical consequences — Bradyarrhythmias and tachyarrhythmias occur in patients with RVMI and contribute to the poor short-term outcome in some patients.

Bradycardia may be due to sinoatrial or atrioventricular nodal dysfunction due to ischemia, activation of cardioinhibitory reflexes, or both. Patients with acute RVMI are at increased risk for both high-grade atrioventricular (AV) block and bradycardia-hypotension without AV block compared to those with inferior MI alone [2,9,26-28]. (See "Conduction abnormalities after myocardial infarction".) The presence of sinus bradycardia (without AV block) and hypotension raises the possibility of cardioinhibitory (Bezold-Jarisch) reflexes arising from stimulation of vagal afferents located in the ischemic LV inferoposterior wall as well as the ischemic RV [29].

Ventricular arrhythmias, including tachycardia and fibrillation, complicate up to one-third of cases of RVMI [9,30]. (See "Clinical features and treatment of ventricular arrhythmias during acute myocardial infarction".)

CLINICAL PRESENTATION — In a patient presenting with an acute myocardial infarction (MI) (particularly ST-elevation MI), the major clinical features of a hemodynamically significant right ventricular infarction (RVMI) include hypotension, elevated jugular venous pressure, and clear lung fields and an electrocardiogram with evidence of an acute inferior MI. ST-elevation >1mm in lead V4R has sensitivity and specificity >90 percent for scintigraphic evidence of RV infarction [31], and approximately 80 percent for echocardiographic evidence of RV dysfunction [32]. The right-sided ST elevation is often transient.

The extent to which RVMI impacts the clinical presentation depends on its size and the relative degree of left ventricular dysfunction. A small RVMI may not lead to hypotension or elevated jugular venous pressure. For these smaller RVMIs, making the diagnosis is less important (than in patients with larger RVMIs), as standard care for (left ventricular) MI will lead to optimal patient care. (See "Overview of the acute management of ST elevation myocardial infarction".)

History and physical examination — The symptoms of RVMI are those common to the broad population of patients with MI: chest pain, nausea, vomiting, diaphoresis, dizziness, and anxiety. However, isolated or predominant RVMI does not cause dyspnea. (See "Initial evaluation and management of suspected acute coronary syndrome in the emergency department", section on 'Clinical presentation'.)

On examination, patients with large RVMI typically present with hypotension (and occasionally shock) and jugular vein distention in the presence of clear lung fields [33]. These physical examination findings in a patient having an MI, although specific, are not sensitive. (See "Clinical manifestations and diagnosis of cardiogenic shock in acute myocardial infarction".)

These findings are in contrast to those found in patients with predominant left ventricular infarction where pulmonary congestion, third or fourth heart sounds, and a new mitral valve murmur may be notable (table 1).

The heart rate in patients with RVMI is generally slower than those with predominant left ventricular MI. Those patients with bradycardia due to vagotonic influences are more likely to manifest other signs of vagal excess, including pallor, diaphoresis, nausea, and vomiting. (See 'Electrical consequences' above.) Tachycardia may be present and is often due to sympathetic discharge related to anxiety or as a compensatory mechanism to raise low cardiac output. (See 'Definitions' above.)

Electrocardiographic features — An electrocardiogram (ECG) should be obtained in all patients with symptoms suspicious for myocardial ischemia. (See "Initial evaluation and management of suspected acute coronary syndrome in the emergency department", section on 'Immediate ED interventions'.)

Any patient with symptoms of an acute coronary syndrome (ACS) and electrocardiographic evidence of inferior wall ischemia or infarction, as evidenced by abnormalities of the ST segment or T wave in leads II, III, and aVF, should have right-sided leads V4R, V5R, and V6R obtained to assess for a possible right ventricular infarct. (See "Electrocardiogram in the diagnosis of myocardial ischemia and infarction", section on 'Inferior and right ventricular MI' and "Electrocardiogram in the diagnosis of myocardial ischemia and infarction", section on 'Inferior MI'.)

The ECG in patients with right ventricular infarction may demonstrate ≥1 mm of doming ST elevation in the right-sided precordial leads V4R to V6R (waveform 1). Right-sided ST elevation, particularly in V4R, is indicative of acute right ventricular injury [9,31,34,35] and correlates closely with occlusion of the proximal right coronary artery [35-37]. In one report of 200 consecutive patients with acute inferior MI, ST elevation in V4R had a sensitivity and specificity for concurrent right ventricular infarction of 88 and 78 percent (respectively), using findings from the results of autopsy, cardiac catheterization, radionuclide imaging, or hemodynamic monitoring as the “gold standard” [9]. Greater ST elevation in lead III than in lead II has been suggested as a predictor of right ventricular infarction, but this finding has not been confirmed [38].

DIAGNOSIS — The diagnosis of right ventricular infarction (RVMI) is strongly suspected when hypotension, raised jugular venous pressure (distended neck veins), and clear lung fields are present in a patient whose 12 lead electrocardiogram has findings of an acute inferior wall infarction as well as ST-elevation in lead V4R. As patients with other diagnoses, such as pericarditis with pericardial tamponade, may present with a similar picture, the diagnosis is usually secured when echocardiography shows RV cavity dilatation and impaired RV free wall motion. Additional testing is rarely needed for diagnostic purposes, but may be helpful for assessment of therapy. The most common circumstance in which additional testing may be necessary is a patient for whom diagnoses of RVMI or pulmonary embolism are still reasonable after echocardiography is performed. (See 'Echocardiography' below and 'Differential diagnosis' below and 'Other imaging studies' below.)

Echocardiography — Urgent echocardiography (often at the bedside), including evaluation for right ventricular infarction, should be performed in patients with an inferior MI and evidence of hemodynamic compromise. In patients without hemodynamic compromise, this test should not delay referral of such patients to the cardiac catheterization laboratory for emergency percutaneous revascularization of the culprit vessel [33,39-43]. (See "Role of echocardiography in acute myocardial infarction".)

Right ventricular size and function and the degree of tricuspid insufficiency are evaluated along with assessment of left-sided structures and function. The major limitation of echocardiography is suboptimal visualization of cardiac structures in some patients.

In studies of patients with either clinical or electrocardiographic features of RVMI and in whom the diagnosis of RVMI was confirmed with autopsy, surgery, radionuclide ventriculography, or hemodynamic monitoring, the most reliable echocardiographic signs of hemodynamically important right ventricular infarction were [41,42]:

- Right ventricular cavity dilatation; cases with right atrial (RA) ischemia may have RA dilation.
- Impaired right ventricular free wall motion (hypokinesis, akinesis, or dyskinesis). The extent of right ventricular wall motion abnormality can vary from affecting only a small region adjacent to the inferior septum and left ventricular inferior segment to affecting a large portion of the right ventricular free wall. Patients with RVMI and hemodynamic compromise are likely to have wall motion abnormalities in a high percent of the right ventricle.
- Diastolic reversed septal curvature, systolic paradoxic septal motion.
- Decreased tricuspid annular plane systolic excursion (TAPSE) and/or reduced right ventricular ejection fraction. (See "Echocardiographic assessment of the right heart", section on 'Tricuspid annular plane systolic excursion (TAPSE)'.)
- Plethora of the inferior vena cava. (See "Echocardiographic evaluation of the atria and appendages", section on 'Vena cava'.)
- Impairment of tissue Doppler measures of right ventricular systolic function [44,45].

The specificity of the findings may be decreased by pre-existent pulmonary disease, such as chronic obstructive lung disease or pulmonary embolism. Pre-existent significant pulmonary artery systolic hypertension (>45 to 50 mmHg) leads to echocardiographic abnormalities of right ventricular structure and function (ie, right ventricular dilatation and tricuspid regurgitation) that mimic some of the findings of RVMI.

Echocardiography is also helpful in identifying cardiac tamponade, which can present similarly, as well as the rare complication of acute ventricular septal rupture complicating RVMI. (See 'Differential diagnosis' below.)

Hemodynamic monitoring — In a minority of patients, due to the limitations of echocardiography, a secure diagnosis of RVMI may not possible. In such patients, placement of a pulmonary artery catheter may provide additional diagnostic information. However, it should be emphasized that an ischemic RV is prone to catheter-induced ventricular arrhythmias and this procedure should be performed with great caution in these patients [30]. (See "Pulmonary artery catheterization: Indications and complications", section on 'Indications'.)

Hemodynamically significant RV infarcts are associated with elevations in right atrial pressure to ≥10 mmHg and a ratio of right atrial pressure to pulmonary capillary wedge pressure to >0.8 (normal mean value <0.6) [41,46,47]. In addition, the cardiac index is decreased. The diastolic filling pressures in the right atrium (RA), right ventricle (RV), and pulmonary capillary (PCW), as well as the LV, may be elevated and equalized. Kussmaul’s sign may be evident in the RA pressure trace (or the jugular venous pulse), reflecting inspiratory augmentation of venous return to a dilated and non-compliant right heart. (See "Examination of the jugular venous pulse", section on 'Kussmaul's sign'.)

As mentioned above, patients with hemodynamically significant RVMI may also have important left ventricular dysfunction, which may prevent the profile presented here from

manifesting.

Once placed, a pulmonary artery catheter may be useful to assess the impact of therapy.

Other imaging studies — Cardiac magnetic resonance imaging (CMR) is considered the standard imaging technique for detailed evaluation of right ventricular structure and function. Contrast-enhanced cardiovascular magnetic resonance is more sensitive for the detection of right ventricular involvement than physical examination, electrocardiography, and echocardiography in patients with an inferior MI [48]. MRI evidence of substantial RV injury (substantial mass of the RV manifesting microvascular obstruction and/or delayed contrast enhancement) may predict adverse outcome [49]. However, at the present time, we do not recommend its use, as there is not yet sufficient evidence to support its use, as it has not been shown to improve patient care in this setting. (See "Clinical utility of cardiovascular magnetic resonance imaging", section on 'Infarct detection and sizing'.)

DIFFERENTIAL DIAGNOSIS — The diagnoses most often confused with right ventricular infarction (RVMI) include pulmonary embolism (PE) (with ST elevation in the right-sided precordial leads caused by "strain"), pericarditis with pericardial tamponade (with ST elevation in many leads, including right sided leads), and anteroseptal MI (ST elevation in leads V1 and V2 may be seen with an RV injury pattern). As discussed above, all patients with suspected acute RVMI and significant hemodynamic compromise should undergo urgent echo, which will typically distinguish among these diagnoses.

Of these, PE and RVMI are most often confused. Both can present with chest pain and findings of clear lung fields and hypotension (including shock) on examination. The nature of the chest pain (ischemic versus pleuritic) may be helpful in making a distinction. The electrocardiogram is usually sufficient to discriminate between the two: ST elevation in the inferior leads is rarely present in patients with PE. Elevation of serum troponin may be present with either diagnosis.

On echocardiography, right ventricular systolic dysfunction may be seen with both diagnoses. Sparing of the right ventricular apex ("McConnell's sign") has been suggested to be specific for a large PE in some reports [50], but not others [51]. We do not consider this finding specific for PE. If the diagnosis remains uncertain, additional testing, such as helical CT scanning or ventilation/perfusion scanning, may be necessary to establish the diagnosis. (See "Diagnosis of acute pulmonary embolism" and "Treatment of acute pulmonary embolism".)

TREATMENT — In general, patients with right ventricular infarction (RVMI) are treated in a manner similar to those with acute ST-elevation MI. This includes the early use of dual oral antiplatelet (aspirin plus a platelet $P2Y_{12}$ receptor blocker), statin therapy, and an anticoagulant. However, medications to improve chest pain such as opioids, nitrates, and beta blockers should be used with caution due to their potential to negatively impact preload (opioids and nitrates) or heart rate and contractility (beta blockers and calcium channel blockers). (See 'Antiischemic drug therapy' below and 'Optimization of right ventricular preload' below.)

Reperfusion, particularly with primary percutaneous coronary intervention, should be initiated as soon as possible. (See "Overview of the acute management of ST elevation myocardial infarction".)

Among patients with RVMI, there is a spectrum of the relative contributions of right and left ventricular dysfunction. (See 'Hemodynamic consequences' above and 'Electrical consequences' above.) Thus, the approach to treatment of abnormal hemodynamics may differ according to the relative contributions. Optimal management requires information obtained from a two-dimensional echocardiogram; some patients will need hemodynamic monitoring. (See 'Echocardiography' above and 'Hemodynamic monitoring' above.)

Therapy in patients who are hypotensive due to predominant RVMI is aimed at improving right ventricular output [2,33]. This is achieved by optimizing right ventricular preload and afterload. In patients who do not respond to these interventions, inotropic support may be necessary to improve right ventricular contractility. In addition, optimization of right ventricular output may require reversing bradycardia or atrioventricular dyssynchrony with appropriate pacing.

Optimization of right ventricular preload — Intravenous fluid (usually isotonic saline) should be given to patients with evidence of low cardiac output (hypotension, hypoperfusion, and a low or normal jugular venous [JVP] pressure) who do not have pulmonary congestion or evidence of right heart failure [33]. This is done to enhance preload and thus improve forward flow out of the right ventricle [2,21,46]. The reported efficacy of this approach is variable, a probable reflection of differences in initial volume status [2].

In most cases, a carefully monitored volume challenge is initiated by infusing aliquots of 200 to 300 mL of normal saline while serially assessing the JVP and blood pressure. An invasive catheter can also be employed, but such instrumentation should not delay emergency revascularization of the infarct-related artery. Once rapid volume infusion results in increases in JVP (or, invasively, the pulmonary capillary wedge pressure) to approximately 15 mmHg without corresponding increases in aortic pressure, further volume expansion is not likely to improve hemodynamics [52].

Nitrates, which are often used to relieve angina, and diuretics, which are given to patients with evidence of pulmonary congestion should be avoided, as they both reduce RV preload. Similarly, opioid drugs may lower preload. An increase in vagal tone caused by insertion of a bladder catheter can acutely decrease preload and lead to cardiogenic shock.

While awaiting the potential benefits of volume loading (or if fluids alone are not sufficient), severe hypotension must be stabilized through administration of inotropic therapy and vasopressors. (See 'Inotropic drugs' below.)

Optimization of right ventricular afterload — Right ventricular output may be further compromised by abnormally high right ventricular afterload, which can occur for a variety of reasons, including:

- Left ventricular dysfunction with elevation in pulmonary venous pressure.
- Hypoxemia from interstitial pulmonary edema, with pulmonary artery vasoconstriction.
- Alpha-adrenergic agonists causing pulmonary vasoconstriction
- Mechanical ventilation with positive end-expiratory pressure.

In patients with predominant RV dysfunction, RV afterload reducing therapy is not indicated and may worsen the hemodynamic profile. In patients with RVMI and significant left ventricular dysfunction, the use of an intraaortic balloon pump, and occasionally afterload reducing agents, may be effective in unloading the left ventricle and subsequently the right ventricle [33]. (See "Prognosis and treatment of cardiogenic shock complicating acute myocardial infarction", section on 'Intraaortic balloon pump' and "Prognosis and treatment of cardiogenic shock complicating acute myocardial infarction", section on 'Vasopressors and inotropes'.)

Optimization of heart rate and AV synchrony — In patients with RVMI, the indications for atropine and temporary pacemakers are similar to the broad population of patients with MI.

The ischemic right ventricle has a relatively fixed stroke volume and therefore right ventricular output is dependent upon heart rate and optimal transport of blood from the right atrium to the RV (referred to as atrioventricular [AV] transport). (See 'Hemodynamic consequences' above.)

As a result, bradyarrhythmias can significantly worsen the hemodynamic status. Atropine may be beneficial to increase heart rate [53], but right ventricular or atrioventricular sequential pacing (to provide an atrial contribution and AV synchrony) may be necessary [2,33,39,54]. However, right ventricular ischemia may lead to suboptimal results from pacing of the right ventricle. The use of atropine and the indications for temporary transvenous pacing in patients with acute MI are discussed elsewhere. (See "Conduction abnormalities after myocardial infarction", section on 'Use of atropine' and "Temporary cardiac pacing", section on 'Temporary pacing in acute myocardial infarction'.)

The development of significant bradycardia with hypotension after reperfusion is not uncommon in patients with inferior MI with RV involvement. Profound bradycardia may be followed by ventricular fibrillation. Some of our authors give atropine before percutaneous coronary intervention (PCI) if there is evidence of vagotonia as suggested by a heart rate <55 beats per minute, even without hypotension.

Inotropic drugs — When fluid resuscitation is insufficient, hypotension should be rapidly corrected with an inotropic agent that also exerts vasoconstrictor effects. Although many vasopressors have been used since the 1940s, few controlled clinical trials have directly compared these agents or documented improved outcomes due to their use [55]. Thus, the manner in which these agents are commonly used largely reflects expert opinion, animal data, and the use of surrogate end points such as tissue oxygenation as a proxy for decreased morbidity and mortality.

Dopamine is the initial agent of choice. The usual starting dose of dopamine is 5 mcg/kg per minute. The dose is titrated up to 15 mcg/kg per minute depending upon the clinical response. Frequent ventricular ectopy and ventricular tachycardia may limit the use of this drug. (See "Use of vasopressors and inotropes", section on 'Dopamine'.)

The usual starting dose of dobutamine is 5 mcg/kg per minute. The dose is titrated up to 20 mcg/kg per minute depending upon the clinical response. Frequent ventricular ectopy and ventricular tachycardia may limit the use of doses above 10 mcg/kg per minute. In addition, since dobutamine decreases peripheral vascular resistance, higher doses may cause hypotension since the cardiac output cannot increase to match the decrease in systemic vascular resistance. (See "Use of vasopressors and inotropes", section on 'Dobutamine'.)

Based on clinical experience, we treat RVMI patients with hypotension refractory to dopamine with mechanical support using an intraaortic balloon pump or direct RV support device. (See 'Mechanical assist devices' below.)

Coronary reperfusion — Early reperfusion using either primary percutaneous coronary intervention or fibrinolytic therapy can preserve both right and left ventricular function as well as reduce mortality and morbidity [7,12,20]. The indications for ar[illegible]se modalities in patients with RVMI are similar to those in patients with left ventricular MI [33,39]. (See "Primary percutaneous coronary intervention in acute ST ele[illegible]nfarction: Determinants of outcome" and "Fibrinolytic therapy in acute ST elevation myocardial infarction: Initiation of therapy".)

Patients in whom reperfusion is achieved typically show a dramat[illegible]he hemodynamic profile within 24 hours and exhibit rapid and complete recovery of RV function. Successfully reperfused patients have dramatically lower incidenc[illegible]dyarrhythmias, tachyarrhythmias, and hypotension, which translates into excellent short- and long-term survival [12].

Mechanical assist devices — An intraaortic balloon pump is useful for the management of patients with cardiogenic shock due to left ventricular dysfunction. Although there are little data on its benefits in shock due to RVMI, we have found it helpful in stabilizing aortic pressure and improving systemic perfusion in some patients and thus may be temporizing in refractory hypotension while performing emergency percutaneous revascularization and subsequently awaiting recovery of RV function. (See 'Hemodynamic consequences' above and "Intraaortic balloon pump counterpulsation" and "Prognosis and treatment of cardiogenic shock complicating acute myocardial infarction", section on 'Intraaortic balloon pump'.)

In cases with RV shock refractory to the above interventions, right ventricular assist devices may be life-saving, as the ischemic RV ultimately tends to recover over time [56]. The rationale for their use is that forward flow into the pulmonary artery may be improved [57]. Both surgically implantable and catheter based right ventricular assist devices are available, but have not been widely used [58].

These devices may provide adequate hemodynamic support to the failing right heart in anticipation of recovery of right heart function. For severe temporary RV failure unresponsive to medical management, FDA-approved surgical right ventricular assist devices (RVADs) have been used as a last resort to stabilize the patient and recover the failing RV. Although significant technical progress has been made in the design of surgical VADs, device- and procedure-related complication and morbidity rates remain substantially high and therefore narrow the scope of their use. The use of a surgical RVAD requires a sternotomy, ventricular or atrial cannulation, and a pulmonary artery cannulation prior to RVAD insertion. The Tandem Heart Percutaneous RV support system has been effective in case reports [56,57]. The Impella RP pump is a new dedicated percutaneous catheter-based percutaneous microaxial pump that is designed for short-term hemodynamic support for the right heart [59]. The Impella RP is advanced through the femoral vein antegrade and positioned across the pulmonary valves, with pump inflow positioned in the inferior vena cava and outflow in the pulmonary artery at the rate of up to 4.0 L/min. The feasibility and safety of this new system is being studied.

Antiischemic drug therapy — Beta blockers and calcium channel blockers, which might be considered as tools to improve ischemia (and in particular, angina), can reduce heart rate and contractility and slow AV conduction.

These drugs should be avoided in patients with RVMI, and in particular, those who are hemodynamically unstable. They can be tried with careful monitoring in those who are stable and have a clear indication [2].

Recommendations of others — Guidelines from the American College of Cardiology/American Heart Association and from the European Society of Cardiology make specific recommendations for the management of patients with RVMI [60,61]. These recommendations are similar to those made in this topic review.

PROGNOSIS — The presence of significant right ventricular involvement in acute myocardial infarction (MI) adversely affects the early outcome. Persistent right ventricular dysfunction adversely affects the late prognosis.

Early prognosis — Prior to the use of primary percutaneous coronary intervention, meta-analyses found that right ventricular involvement in patients with an acute inferior MI was associated with a worse in-hospital outcome due primarily to persistent hypotension and arrhythmias [9,12,20,62-64]. In a meta-analysis of six studies, which included 1198 patients, the presence of right ventricular involvement (compared to no RV involvement) was associated with a higher incidence of short-term death (odds ratio [OR] 3.2, 95% CI 2.4-4.1), cardiogenic shock (OR 3.2, 95% CI 2.4-3.5), sustained ventricular tachyarrhythmias (OR 2.7, 95% CI 2.1-3.5), and advanced atrioventricular block (OR 3.4, 95% CI 2.7-4.2) [63]. The increase in mortality appeared to be related to the presence of right ventricular involvement and not to infarct size.

Among patients who are diagnosed with right ventricular infarction (RVMI) and cardiogenic shock, in-hospital and 30-day mortality have been reported to be 53 and 23 percent, respectively [65,66]. (See "Prognosis and treatment of cardiogenic shock complicating acute myocardial infarction".)

Long-term prognosis — The prognosis of patients with RVMI has dramatically improved with the advent of routinely applied emergency percutaneous revascularization [12]. The right ventricle typically recovers much of its function [7,8,13,16,46].

In those who survive RVMI, the long-term prognosis is primarily determined by the extent of left ventricular involvement. This is particularly true in patients with anterior MIs in whom the right ventricle accounts for a much smaller proportion of total infarct size than inferior MIs (7 versus 28 percent) [1]. Nearly complete recovery of RV function has been shown to occur in 62 to 82 percent of patients within the first few months [7,8]. Chronic right heart failure attributable only to right ventricular infarction is rare. Even in those without successful reperfusion, if they survive, RV function returns to near normal at rest and with exercise [14].

Over the long term, a persistent reduction in right ventricular function appears to be associated with a worse long-term prognosis [8]. This was illustrated in a study of 147 patients more than 30 days after MI (mean age of infarct 6.7 years) in whom the right ventricular ejection fraction (RVEF) was assessed with contrast enhanced cardiovascular magnetic resonance imaging [67]. During a mean follow-up of 17 months, patients with an RVEF <40 percent had a significantly higher mortality compared to those with an RVEF >40 percent (adjusted hazard ratio 2.9).

SUMMARY AND RECOMMENDATIONS

- Right ventricular myocardial infarction (RVMI) commonly accompanies acute infarction of the inferior wall of the left ventricle, occurring in more than one-third of such cases. The majority of right ventricular infarcts result from occlusion of the proximal right coronary artery. (See 'Introduction' above.)
- The diagnosis of RVMI can be strongly suspected when hypotension, raised jugular venous pressure (distended neck veins), and clear lung fields are present in a patient whose 12-lead electrocardiogram has findings of an inferior wall infarction as well as ST-elevation in lead V4R.

  As patients with other diagnoses, such as pericarditis with pericardial tamponade or pulmonary embolism, may present with a similar picture, the diagnosis is usually secured when echocardiography shows RV cavity dilatation and impaired RV free wall motion. For those uncommon patients in whom either RVMI or pulmonary embolism remains a diagnostic possibility after echocardiography, additional testing, such as helical computed tomography (CT) scanning or ventilation/perfusion scanning, may be necessary to establish the diagnosis. (See 'Clinical presentation' above and 'Diagnosis' above and 'Differential diagnosis' above.)
- In general, patients with RVMI are treated in a manner similar to those with acute ST-elevation MI. This includes the early use of dual oral antiplatelet (aspirin plus a platelet $P2Y_{12}$ receptor blocker) and statin therapy as well as anticoagulant. However, drugs that lower preload (eg, nitrates or diuretics), slow heart rate (eg, beta blockers), or decrease contractility (eg, calcium channel blockers) need to be used with caution. Reperfusion, particularly with primary percutaneous coronary intervention, should be initiated as soon as possible. (See 'Treatment' above.)
- In patients with RVMI:
  - For patients with evidence of low cardiac output, clear lung fields, and normal or low jugular venous pressure, small boluses of normal saline should be administered. Medications that decrease preload, such as nitrates or opioids, should be avoided. (See 'Optimization of right ventricular preload' above.)
  - For patients with persistent hypotension after an attempt to optimize RV preload with normal saline boluses, we suggest adding dopamine (**Grade 2C**). The usual starting dose is 5 mcg/kg per minute. (See 'Inotropic drugs' above.)
  - Patients with predominant RVMI do not benefit from afterload reducing therapy with either an intraaortic balloon pump (IABP) or vasodilating agents. However, some patients have increased RV afterload due to left ventricular dysfunction and their hemodynamic profile is determined by biventricular dysfunction. In this setting, the use of an intraaortic balloon pump or, in uncommon cases vasodilating agents, may improve the hemodynamic profile. (See 'Optimization of right ventricular afterload' above.)
  - As bradycardia and atrioventricular dyssynchrony can rapidly worsen the hemodynamic status of patients with a right ventricular MI, attempts to correct these electrical abnormalities should be instituted early. The indications for the use of atropine and temporary transvenous pacing are similar to those for the broad population of patients with MI. (See 'Optimization of heart rate and AV synchrony' above.)

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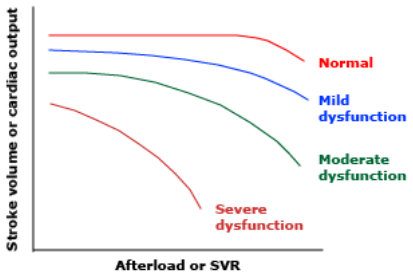
Topic 75 Version 11.0

GRAPHICS

Effect of increasing afterload on cardiac function



Curves relating stroke volume or cardiac to afterload or systemic vascular resistance (SVR) in normal subjects and those with heart failure and increasing degrees of ventricular dysfunction. Increasing afterload has little acute effect in normal subjects but becomes progressively more limiting on cardiac output in HF. Even though the effect is small with mild HF, lowering afterload is still important over the long-term because it slows the rate of loss of myocardial function.

Graphic 55742 Version 1.0

Differences between left and right ventricular myocardial infarction

	Left ventricular MI	Right ventricular MI
Clinical features	Pulmonary congestion	Clear lung fields
	Third and fourth heart sounds	Right-sided third heart sound
	New mitral regurgitation	New tricuspid regurgitation
		Hypotension with distended neck veins
Electrocardiogram	ST elevation in standard leads	ST elevation in V4R
		Commonly associated with inferior MI
		Frequent atrioventricular block
Hemodynamic findings	Increased pulmonary capillary wedge pressure (PCWP)	Right atrial pressure (RAP) ≥10 mmHg
		RAP:PCWP ≥0.8
Specific management	Fluid restriction	Fluid resuscitation
	Preload and afterload reduction	Avoid preload reduction
	Reperfusion therapy	Reperfusion therapy
	Inotropic agents, if necessary	Inotropic agents, if necessary

Graphic 62368 Version 1.0

ECG of acute inferior and right ventricular myocardial infarction

Electrocardiogram shows Q waves and prominent doming ST segment elevation in II, III, and aVF, findings which are characteristic of an acute inferior myocardial infarction. ST elevation in the right precordial leads - V4R, V5R, and V6R - indicates right ventricular involvement as well (arrows). The ST depressions in leads I and aVL represent reciprocal changes.

Courtesy of Ary Goldberger, MD.

Graphic 82739 Version 3.0

Normal ECG

Normal electrocardiogram showing normal sinus rhythm at a rate of 75 beats/min, a PR interval of 0.14 sec, a QRS interval of 0.10 sec, and a QRS axis of approximately 75°.

Courtesy of Ary Goldberger, MD.

Graphic 76183 Version 3.0